

results from reinfection. This is caused by the prolonged excretion of the organism among untreated children, lasting up to seven weeks.⁵ On the other hand, stool cultures of all treated patients yielded negative results within 48 hours.⁵ Our patients indeed showed that simultaneous treatment is effective in preventing reinfection as it caused concomitant eradication of the infection in all the children.

It is concluded that in a nursery with multiple cases of campylobacter enteritis stool cultures should be taken from all children, including those who are asymptomatic at that time, because it is necessary to treat *simultaneously* all children whose cultures yield positive results. This approach is an effective method for eradicating the infection.

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Commentary

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It is often forgotten that antimicrobial drugs can sometimes be used to control the spread of gut infection. This 'epidemiological' use has a chequered history. Most often non-absorbable antibiotics, such as neomycin, have been employed to blanket an outbreak, especially of *Escherichia coli* gastroenteritis, and with variable results. Sometimes the outbreak was apparently controlled and at other times the attempt failed completely; certainly, antibiotic prophylaxis should not be used in this way unless combined with a rigorous attempt to tighten up normal hygienic methods of cross infection control.

This description of the control of campylobacter gastroenteritis in a day care centre makes a useful point. Features of this infection that perhaps make for a favourable outcome are:

(1) Erythromycin certainly seems to render stool cultures negative very quickly, usually within 48 hours of starting the drug (their references 1 and 6).

(2) The duration of carriage may be quite long without treatment, although 90% of patients do become negative spontaneously within two months.

(3) Campylobacter diarrhoea, although usually short lived, can be prolonged or recurrent, and it is patients with diarrhoea who pose most risk to their contacts. Although eight of the 12 contacts with positive stools were asymptomatic at the time of sampling, all of them had had recent diarrhoea.

Co-trimoxazole red cell aplasia in leukaemia

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SUMMARY A 4 year old boy with acute lymphoblastic leukaemia developed a pure red cell aplasia 13 months after entering remission and while on maintenance chemotherapy. Co-trimoxazole was also being administered for prophylaxis against *Pneumocystis carinii* infection. When co-trimoxazole was stopped the red cell aplasia resolved.

thoprim) provides effective prophylaxis against *Pneumocystis carinii* infection¹ and is therefore commonly used during treatment for childhood acute lymphoblastic leukaemia. Haematological toxicity, particularly neutropenia and thrombocytopenia, has been described with co-trimoxazole,^{2,3} but selective aplasia of the bone marrow erythroid series is extremely rare and has not previously been reported in acute lymphoblastic leukaemia. We report a case of pure red cell aplasia that occurred in a child on maintenance treatment for acute lym-

Co-trimoxazole (sulphamethoxazole and trime-

phoblastic leukaemia and that completely resolved when treatment with co-trimoxazole was stopped.

Case report

A 4 year old boy developed acute lymphoblastic leukaemia in July 1983 and remission was promptly obtained using vincristine, prednisone, adriamycin, and L-asparaginase. In September 1983, after consolidation treatment, maintenance treatment with three weekly sequences of vincristine, prednisone, 6-mercaptopurine, and methotrexate was given. Treatment with co-trimoxazole (as trimethoprim 40 mg daily) was begun for pneumocystis prophylaxis once remission had been obtained.

Haemoglobin concentrations remained satisfactory during early maintenance treatment and a bone marrow examination in February 1984 (because of persistent neutropenia) showed hyperplastic megaloblastic erythropoiesis but no evidence of acute lymphoblastic leukaemia.

After 13 months in remission (November 1984) the haemoglobin fell 64 g/l over a five week period (Figure). The red cells were normochromic and normocytic (mean corpuscular volume 88 fl, mean

corpuscular haemoglobin 31.1 pg, and mean corpuscular haemoglobin 354 g/l). The reticulocyte count was less than 0.1% and white cell and platelet counts yielded normal results. A previous reticulocyte count of 3% had been recorded six months earlier. Serum folate and vitamin B₁₂ concentrations were normal. One unit of packed red cells was transfused but the subsequent rise in haemoglobin was only transient. Transfusions were required at roughly monthly intervals to maintain an adequate haemoglobin. The reticulocyte count always remained below 0.1%. White cell and platelet concentrations remained unchanged throughout this period compared with previously. Bone marrow examination in March 1985 showed markedly hypoplastic erythropoiesis with only 4% erythroblasts. An occasional cell was megaloblastic. Serum folate and vitamin B₁₂ concentrations were again normal. White cell and platelet precursor series were normal, with no evidence of leukaemic relapse. A further two units of packed cells were transfused and co-trimoxazole was stopped in case this was responsible for the red cell aplasia.

Over the next six weeks the patient's haemoglobin remained stable, ranging between 116 and 127 g/l.

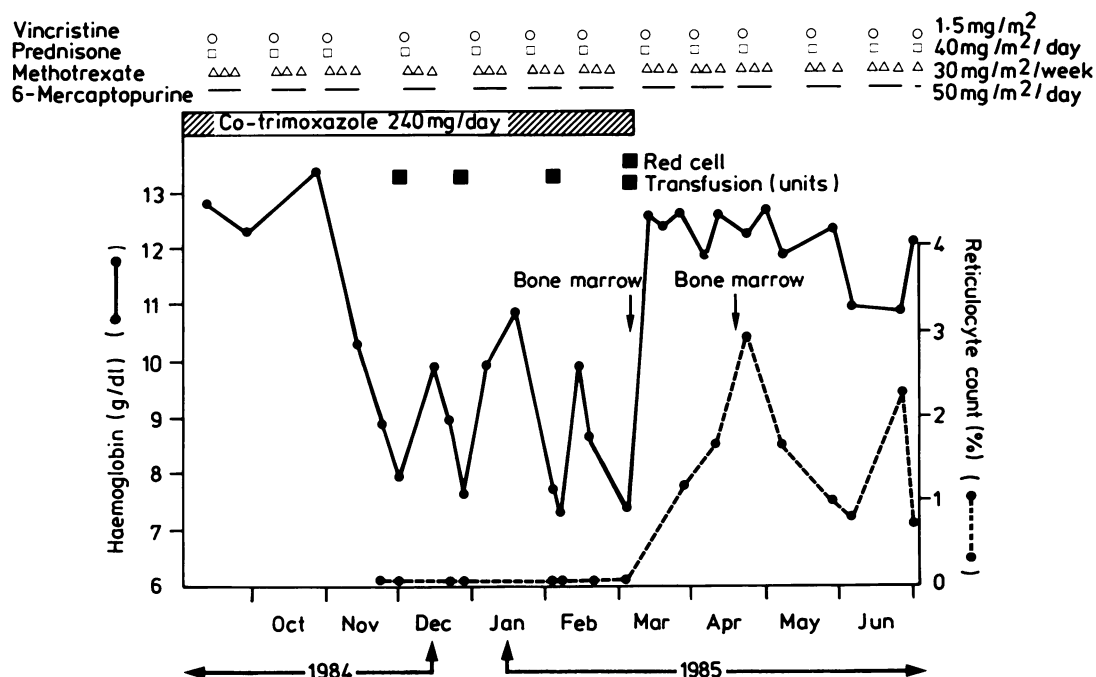


Figure Treatment regimen in a case of red cell aplasia. Bone marrow examinations and packed red cell transfusions are shown.

The reticulocyte count rapidly increased to a peak of 3.7% in mid-April. Repeat bone marrow examination six weeks after stopping of treatment with co-trimoxazole showed complete resolution of red cell aplasia with 28% erythroblasts, indicating normal erythropoiesis. The haemoglobin and reticulocyte counts have continued to remain at satisfactory levels and no further blood transfusions have been required. Co-trimoxazole has not been restarted.

Discussion

Selective erythroid aplasia has been reported in two children receiving combination therapy for acute lymphoblastic leukaemia,⁴ being reversible on stopping treatment in one patient. Neither patient was exposed to co-trimoxazole. Erythroid hypoplasia developed in an adult patient with Crohn's disease on long term treatment with co-trimoxazole.⁵ Drysdale has reported a child with acute lymphoblastic leukaemia, on similar treatment to our patient, who developed transfusion dependent anaemia when co-trimoxazole prophylaxis was begun.⁶ This patient also took sodium valproate for epilepsy and this drug was thought to be contributory. A red cell aplasia was not proved by bone marrow examination.

Our patient's red cell aplasia developed after 13 months of treatment with co-trimoxazole. Maintenance chemotherapy remained unchanged before, during, and after the episode. There was no exposure to other drugs over the entire period. Complete

resolution of the bone marrow abnormality occurred after stopping treatment with co-trimoxazole.

We believe that this is the first reported case of red cell aplasia attributable to treatment with co-trimoxazole in a child with acute lymphoblastic leukaemia. Red cell aplasia induced by co-trimoxazole should be considered in this disease should unexplained anaemia develop.

We thank Dr MEJ Beard, haematologist, for his help with this case.

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